

**МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ**



МАТЕРІАЛИ

**106-ї підсумкової науково-практичної конференції
з міжнародною участю
професорсько-викладацького колективу
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THE ROLE OF INFLAMMATORY MARKERS IN THE FORMATION OF SUBCLINICAL ATHEROSCLEROSIS

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Introduction. The proposed 2018 definition of clinical conditions in cardiology, which can serve as a manifestation of subclinical atherosclerosis, including asymptomatic patients at risk for coronary heart disease, atypical course, changing the development of acute coronary syndrome, long preclinical period against the background of confirmed coronary atherosclerosis can cause some changes in diagnostic and treatment strategy according to the latest European guidelines.

The aim of the study. To investigate the influence of Pregnancy-Associated Plasma Protein A (PAPP-A) and C-Reactive Protein (CRP) levels on the development of subclinical atherosclerosis. Key parameters assessed include the rate of change in carotid intima-media thickness (CIM), total ejection fraction, end-systolic volume, total cholesterol and exercise tolerance. Additionally, a comparison was made between the initial biomarker levels and changes observed in the treatment group (n=23) receiving statins and metabolic therapy (trimetazidine and magnesium B6).

Materials and methods. A total of 67 patients were examined, divided into two groups based on clinical manifestations: subclinical atherosclerosis and atypical presentation, with a focus on differential diagnosis across vegetative-vascular dystonia, coronary syndrome X, and stable angina (stress I-II functional class). Biomarker levels, including Pregnancy-Associated Plasma Protein A (PAPP-A) and C-Reactive Protein (CRP), were assessed. A comprehensive clinical and functional evaluation of all patients was conducted using various methods, including electrocardiography (ECG), echocardiography, treadmill testing, and blood tests (including enzyme-linked immunosorbent assay, ELISA).

Results. CIM indication decreased during treatment and surveillance in the general group (n = 67) (p <0,05) and the distribution of PAPP-A $\geq 4,12$ mIU/L (p <0,002), and observations determined initial increase in CIM by distribution PAPP-A $\geq 4,12$ mIU/L (p <0,001), which were stored and during treatment in the total group (n = 67) in the distribution of medium-sized CMMs for PAPP-A were in the treatment $\geq 4,12$ mIU/L (p <0,01). In the group before /after treatment (n = 23) there was a decrease of CIM during treatment in the group general (p <0,02), with a tendency to decrease CIM in the group where enlarged PAPP-A $\geq 4,48$ mIU/L (p >0,05) and reduced PAPP-A <4,48 mIU/L (p >0,05), and subclinical atherosclerosis (n = 46) registered a decrease CIM in the treatment group reduced PAPP-A (<4,54 mIU/L, p <0,01), but not in the group of increased PAPP-A ($\geq 4,5$ mIU/L, p >0,1). In our own study we highlight a significant decrease in the sum of CIM based content CRP in the group overall (n=67) during treatment (p <0,02) and at distribution of CRP $\geq 12,47$ mg/l was recorded a CIM reduction (p <0,005). The initial increase in CIM, which further decreases significantly in the treatment group (n=23) for the distribution of CRP <17, 11 \geq mg/dL (p <0,02), also significantly reduce CIM consistent for CRP in the treatment group PSA $\geq 12,47$ mg/L (p <0,005), as well as in atherosclerosis group for CRP (<16,55 \geq mg/l) with decreasing rate CIM (p <0,05).

Conclusions. CIM index decreased during treatment and surveillance in the general group (n=67) (p <0,05) and the distribution of PAPP-A $\geq 4,12$ mIU/ L (p <0,002), for a specified output increase by CIM distribution PAPP-A $\geq 4,12$ mIU/L (p <0,001), which were stored and during treatment in the total group (n=67) in the distribution of average CMM for PAPP-A in the treatment of $\geq 4,12$ mIU/L (p <0,01). The initial increase in CIM, which further decreases significantly in the treatment group (n=23 for the distribution of CRP <17, 11 \geq mg/l (p <0,02), also significantly reduce CIM consistent for CRP in the treatment group PSA $\geq 12,47$ mg/l (p <0,005), as well as in atherosclerosis group for CRP (<16,55 \geq mg/l) with decreasing rate CIM (p <0,05).